brain type of non-insulin dependent diabetes mellitus? A challenging hypothesis. J. Neural Transm. 105, 415-422, 1998). It has been suggested that intracerebroventricular insulin enhances memory in a passive-avoidance task [Park, C. P., Seeley, R. J., Craft, S. and Woods S. C. (2000) Intracerebroventricular insulin enhances memory in a passive avoidance task. Physiol. Behav. 68, 509-514]. Insulin receptor density and tyrosine kinase activity in the sporadic Alzheimer's disease (SDAT) was known to be significantly decreased [Frolich, L., Blum-degen, D., Bernstein, H. G., Engelsberger, S., Humrich, J., Laufer, S., Muschner, D., Thalheimer, A., Turk, A., Hoyer, S., Zochling, R., Boissl, K. W., Jellinger, K., and Piederer, P. Brain insulin and insulin receptors in aging and sporadic Alzheimer's disease. J. Neural Transm. 105, 423-438, 1998]. Interestingly, tyrosine phosphorylation of the hippocampal insulin receptor has been shown to play an essential role in spatial memory formation [Zhao, W., Chen, H., Xu, H., Moore, E., Meiri, N., Quon, M. J., Alkon, D. L. (1999) Brain insulin receptors and spatial memory. J. Biol. Chem. 274, 34893-34902, 1999].--

a1

Please add a NEW paragraph numbered [0010] after paragraph [0009] as follows:

--Recently, it has been found that ERK (Extracellular signal-Regulated Kinase or MAPK) I and II, which are important downstream

a2

signaling mediators of the insulin receptor, are implicated in memory and learning [Thiels, E, Klann, E. Extracellular signal-regulated kinase, synaptic plasticity, and memory. Rev. Neurosci. 12, 327-345, 2001; Sweat J.D. The neuronal MAP kinase cascade: a biochemical signal integration system subserving synaptic plasticity and memore. J. Neurochem. 76, 1-10, 2001]. It has been also demonstrated that rats subjected to avoidance learning showed significant and specific increases in the activated forms of ERK I and II in the rat hippocampus [Cammarota, M., Bevilaqua, L.R.M., Ardenghi, P., Paratcha, G., de Stein, M.L., Iaqueirdo, I., Medina, J.H. Learning-associated activation of nuclear MAPK, CREB and Elk-1, along with Fos production, in the rat hoppocampus after a one-trial avoidance learning; abolition by NMDA receptor blockade. Mol. Brain Res. 76, 36-46, 2000]. Taken together, insulin receptor and ERK I/II activators could be used for memory enhancement in addition to cholinesterase inhibitors.

Please replace Paragraph[0072] with the following rewritten paragraph:

--The test was basically performed according to the step-through method described by Jarvik and Kopp [Jarvik, M. E. and Kopp, R. An improved one-trial passive avoidance learning situation. Pschol. Rep. 21, 221-224, 1967]. The Gemini Avoidance System (SD Instruments) was used for this experiments. The

apparatus consists of a two-compartment acrylic box with a lightened compartment connected to a darkened one by an automatic guillotine door. Mice were placed in the lighted box for 300 sec. Then, the guillotine door was open. Mice, as soon as they entered the dark compartment, received a punishing electrical shock (0.3 mA, 1 sec). The latency times for entering the dark compartment were measured in the training test and after 24 hr in the retention test. The maximum entry latency allowed in the retention session was 500 sec. Fraction 1, 2 or 4 (10 mg/kg/day, P.O.) was administered once a day for three days and tested for the passive avoidance test.--

Please replace paragraph[0078] with the following rewritten paragraph:

--Male Sprague Dawley rats were decapitated after 60 min. following the administration of AR extracts and subjected to the isolation of hippocampus on 4C.. Hippocampal homogenates were prepared as described earlier with some modifications [Zhao, W., Chen, H., Xu, H., Moore, E., Meiri, N., Quon, M. J., Alkon, D. L., Insulin receptors and spatial memory. J. Biol. Chem. 274, 34893-34902, 1999]. The isolated hippocampus was resuspended with buffer A containing 50 mM Tris HCl, pH 7.4, 1 mM EDTA, 1 mM EGTA, 150 mM NaCl, 1% Triton X-100, 0.5 mM PMSF, 1 mM Na_3VO_4, 1ug/ml of leupeptin and aprotinin and subjected to homogenization with a Potter-

Elvehjem homogenizer. The lysates were then spun at 10,000 x g for 20 min and the supernatant were subjected to protein assay and saved at 70°C.--

Please replace paragraph[0083] with the following rewritten paragraph:

--Equal amount of hippocampal proteins were applied to SDS polyacrylamide gel. Electrotransfer of proteins from the gels to nitrocellulose paper (Schleicher & Schuell) was carried out for 1 hr at 100 V (constant) as described by Towbin et al. [Towbin H., Staehelin, J., Gordon, J. Electric transfer of proteins from polyacrylamide gels to nitrocellulose sheets: procedure and some applications. Proc. Natl. Acad. Aci. USA 76, 4350-4354, 1979].The filter papers were preincubated for 1 hr at 23 C with PBS containing 0.1% Tween 20 and 3% bovine serum albumin and washed with PBS containing 0.1% Tween 20 three times for 10 min each. The blots were probed with pTyr or pERK antibodies for 1 hr at 23 C. The blots were then incubated with HRP-conjugated anti-rabbit IgG for 30 min and washed with PBS containing Tween 20 five times for 10 min each. The detection of immobilized specific antigens was carried out by ECL (NEN). --

Please replace paragraph[0088] with the following rewritten paragraph:

--Male SD rats were dosed p.o. with vehicle or fractions of AR extract. The rats were decapitated after 60 min, brains rapidly removed, hippocampus and corpora striata dissected free, weighed and homogenized as described above. Cholinesterase activity was measured as described by Ellman et al [Ellman, G. L., Courtney, K. D., Andres, V., Featherstone, R. M. A new and rapid colorimetric determination of acetylcholinesterase activity. Biochem. Pharmacol. 7, 88-95.1961]. Briefly, 3 ml of buffer I (100 mM phosphate, pH 8.0), 0.2 ml of 75 mM acetylthiocholine iodide and 0.1 ml of buffered Ellman's reagent (DTNB 10 mM, NaHCO3 15 mM) were mixed and allowed to incubate for 10 min at 25°C. Then, 20 ml of enzyme sample was added and absorbance was measured at 30 sec intervals. The percent inhibition was calculated by comparison with the enzyme activity of the vehicle control group.--

IN THE CLAIMS:

Please amend the following claims:

1. (Amended) A composition containing Asiasari Radix extracts having at least two therapeutically effective agents therein for protecting brain cells against damage caused by excitatory amino acids and oxidative stresses.

11. (Amended) The composition of Claim 1, wherein said the Asiasari Radix extracts are obtained by the following sequential fractionation procedure: